Skeletal Radiol (1995) 24:160–162

Skeletal Radiology

Polyostotic fibrous dysplasia with severe pathologic compression fracture of L2

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Clinical information

This female patient first presented aged 21 years with a 2.5-year history of gradual onset of increasing back pain. She had previously been in excellent health. No other significant symptoms were noted at that time. Conventional radiographs of the lumbar spine (Fig. 1) showed a pathologic compression fracture with mild anterior wedging of L2, leading to a 30° kyphotic angulation deformity at this level. There was expansion of the vertebral body but without interruption of the thinned cortex. The matrix was non-mineralized. A radionuclide bone scan demonstrated increased activity involving multiple bony structures. Magnetic resonance (MR) imaging of the lumbar spine (Fig. 2) showed abnormally low signal on both T1- and T2-weighted sequences within the entire marrow space of the L2 and L5 vertebral bodies. Biopsy of the L2 vertebral body was performed, but no other action was taken.

Two years later the patient, now aged 23 years, again presented with worsening back pain as well as new radicular symptoms. Lumbar spine radiographs showed further collapse of the L2 vertebral body (Fig. 3) with a 60° kyphotic deformity. MR imaging (Fig. 4) demonstrated interval further collapse of the L2 vertebral body as compared to the MR study 2 years earlier, now with associated narrowing of the spinal canal. Anterior vertebral fusion with a strut allograft and posterior fusion with spinal rods with pedicle screws was performed. The resected vertebral fragments were submitted for pathological examination.

Microscopic examination of specimens from both the initial biopsy and subsequent resection (Fig. 5) demonstrated irregularly shaped spicules of woven bone set in a bland fibrous stroma. No osteoblastic rimming was seen. These histological findings were diagnostic of fibrous dysplasia.

Discussion

Fibrous dysplasia is a disorder of bony structures characterized by fi-

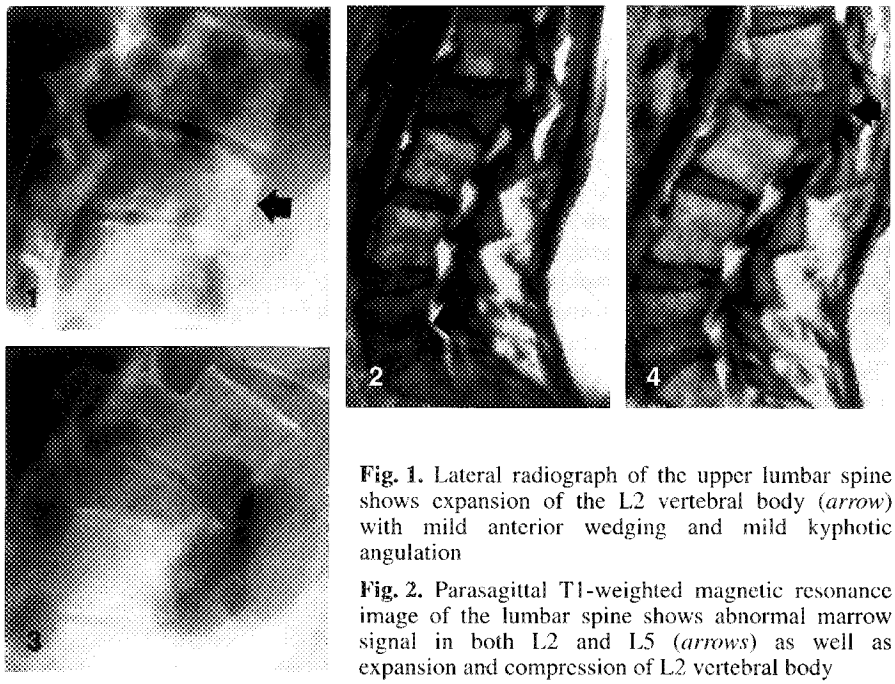

Fig. 1. Lateral radiograph of the upper lumbar spine shows expansion of the L2 vertebral body (*arrow*) with mild anterior wedging and mild kyphotic angulation

Fig. 2. Parasagittal T1-weighted magnetic resonance image of the lumbar spine shows abnormal marrow signal in both L2 and L5 (*arrows*) as well as expansion and compression of L2 vertebral body

Fig. 3. Lateral radiograph obtained 2 years later demonstrates severe progression of both pathologic compression fracture and kyphotic angulation

Fig. 4. Parasagittal T1-weighted magnetic resonance image of the lumbar spine obtained at the same time as Fig. 3 shows the severe collapse and angulation as well as compromise of the spinal canal (*arrow*)

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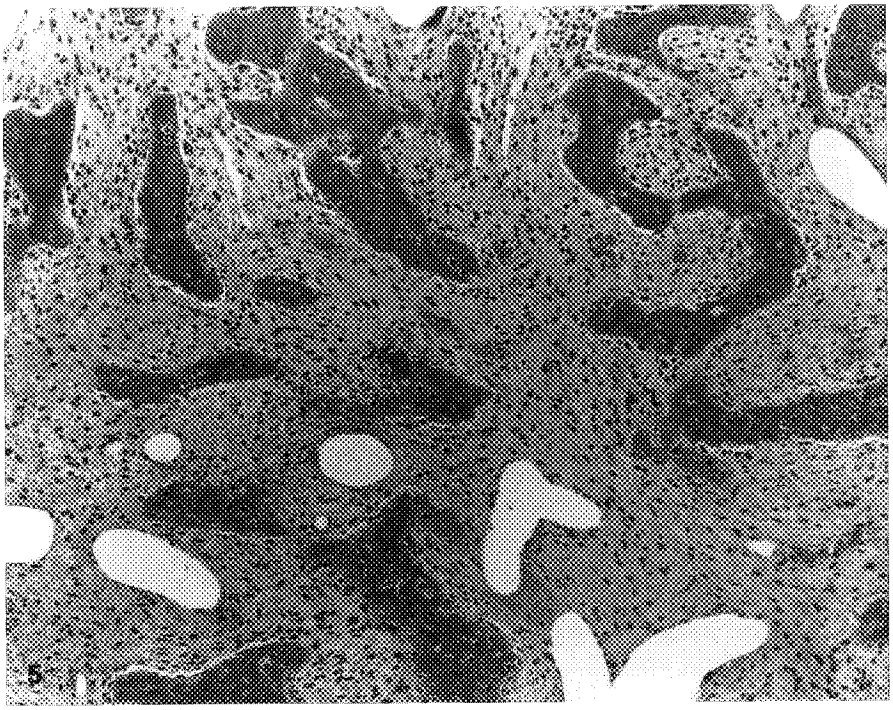

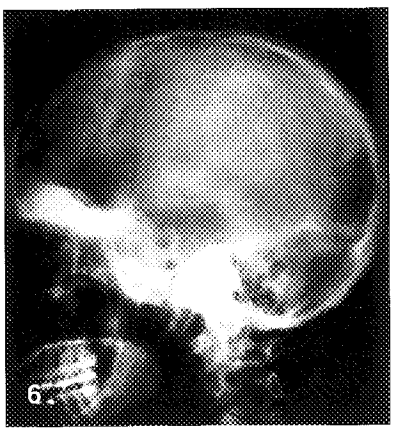

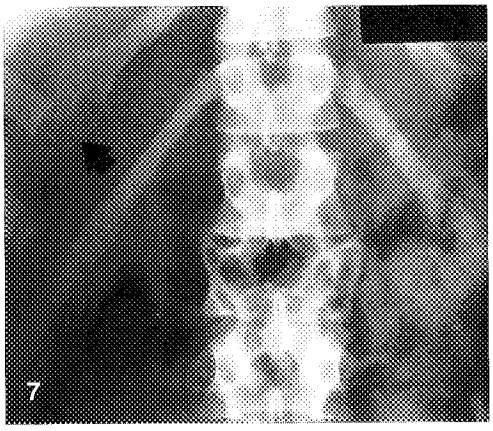

Fig. 5. Histologic section of the initial vertebral bone biopsy. Haphazardly arranged and irregularly shaped bone trabeculae are set in a paucicellular bland fibrous stroma

Fig. 6. Lateral radiograph of the skull shows bony expansion and increased density of the skull base, characteristic of fibrous dysplasia

Fig. 7. Anteroposterior radiograph of the thoracolumbar junction, obtained at the same time as Fig. 1, shows bony expansion of both the L2 vertebral body and the right 11th rib (*arrow*)

brous replacement of normal medullary bone due to dysfunctional bone forming elements. It accounts for approximately 7% of all benign bone lesions [1].

Fibrous dysplasia is more commonly monostotic (85%) than polyostotic (15%). Spinal involvement is rare with the monostotic disease, but has been reported to involve the cervical, thoracic, or lumbar spine [2–4]. Polyostotic fibrous dysplasia more commonly involves the spine, with an incidence ranging from 7% in the cervical spine [5] to 14% in the lumbar spine [6]. In the majority of cases these lesions are asymptomatic and require no treatment [2]. In the present case, however, treatment was necessitated by the severe and progressive collapse resulting in a severe kyphotic deformity and neurologic symptoms.

No reports of significant lumbar spine involvement by pathologic fracture in the absence of significant trauma have been published previously. In the cervical spine, previous case reports of multilevel vertebral body involvement have documented both severe deformity and neurologic dysfunction [7]. Post-traumatic pathologic fracture of the odontoid process has also been reported [8].

The present case demonstrates characteristic radiographic (Figs. 6, 7), radionuclide, and MRI (Figs. 2, 4) findings of fibrous dysplasia. On the initial study, the L2 vertebral body showed expansion. Although not evident in this case, the posterior elements are frequently reported to be involved in fibrous dysplasia of the spine [9].

Increased activity on bone scan has been attributed to greater than normal vascularity within fibrous dysplasia lesions [10]. These studies are more sensitive for detection of fibrous dysplasia than are radiographic examinations [11], and in the present case revealed involvement of the L5 vertebral body which was not identified on the radiographs.

MRI findings vary depending upon the fibrous, cartilaginous, and sometimes hemorrhagic components of fibrous dysplasia [12]. Intermediate signal is most commonly seen on T1-weighted images, while T2-weighted images show low signal for predominantly fibrous lesions, high signal for high cartilage content le-

sions and mixed signal for lesions containing both [1]. MRI also demonstrates the extent of vertebral body involvement and, as in the present case, neural canal involvement.

The radiographic differential diagnosis includes hemangioma, aneurysmal bone cyst, and giant cell tumor.

In *summary,* a case of a progressive pathologic compression fracture of L2 due to polyostotic fibrous dysplasia is presented. Such unusually severe involvement, requiring decompression and surgical fusion, in the absence of significant recent or remote trauma, has not previously been reported.

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